Amendments to the Claims

1. (Previously presented) A method of screening an agent to determine its usefulness in treating a condition characterised by abnormal body weight or eating dysfunction, the method comprising:

(a) establishing a paradigm in which at least one protein is differentially expressed in relevant tissue from, or representative of, subjects having differential levels of body weight or eating dysfunction;

(b) obtaining a sample of relevant tissue taken from, or representative of, a subject having body weight or eating disorders, who or which has been treated with the agent being screened;

(c) determining the presence, absence or degree or expression of the differentially expressed protein or proteins in the tissue from, or representative of, the treated subjects; and

(d) selecting or rejecting the agent according to the extent to which it changes the expression of the at least one differentially expressed protein in the treated subject having body weight or eating disorders.

2. (Previously presented) The method of claim 1, wherein the agent is selected if it converts the expression of the at least one differentially expressed protein towards that of a subject having a more normal body weight or eating behaviour.

3. (Previously presented) The method of claim 1, wherein the agent is selected if it converts the expression of the at least one differentially expressed protein to that of the normal subject.

4. (Previously presented) The method of claim 1, wherein the body weight or eating disorder is a result of at least one of (i) a disorder which causes an increase in body weight or (ii) a disorder which is associated with an excess food consumption.

5. (Previously presented) The method of claim 1, wherein the paradigm is based on tissue from obese subjects and normal subjects.

6. (Previously presented) The method of claim 1, wherein the paradigm is based on a comparison of subcutaneous and omental adipose tissue from the same individuals.

7. (Previously presented) A method of claim 1, wherein the body weight or eating disorder is a result of at least one of (i) a disorder which causes a reduction in body weight or (ii) a disorder which is associated with a low food intake.

8. (Original) A method of claim 7, wherein the paradigm is based on tissue from subjects with anorexia nervosa or bulimia or AIDS or cancer and normal subjects.

9. (Previously presented) The method of claim 1, wherein the paradigm is based on animals which are models of obesity as a result of a genetic mutation together with lean littermates.

10. (Original) The method of claim 1, wherein the paradigm si based on animals in which obesity is induced or exacerbated by dietary treatment.

11. (Previously presented) The method of claim 1, wherein the paradigm is based on lean and obese animals obtained by a selective breeding program from a common stock.

12. (Previously presented) The method of claim 1, wherein the paradigm is based on desert rodents, which develop obesity on normal laboratory diets.

13. (Original) The method of claim 1, wherein differential levels of obesity occur in apparently similar animals in which it is attempted to induce obesity by dietary modification.

14. (Previously presented) The method of claim 1, wherein in the paradigm, the subjects having differential levels of body weight or function comprise normal subjects and obese subjects.

15. (Previously presented) A method of claim 1, wherein in the paradigm, the subjects having differential levels of body weight comprise obese subjects and body weight reduced previously obese subjects or obese subjects and subjects who resisted the development of obesity.

16. (Previously presented) The method of claim 1, wherein in the paradigm, the subjects having differential levels of body weight comprise normal subjects and subjects having a below normal body weight.

17. (Original) The method of claim 15, wherein thebody weight of a subject is reduced by treatment with a drug, dietary restriction or exercise.

18. (Original) The method of claim 17, wherein the drug is a thermogenic drug.

19. (Original) The method of claim 18, wherein the thermogenic drug is a b_3-adrenoceptor agonist or leptin.

20. (Original) The method of claim 17, wherein the drug is an anorexic drug.

21. (Original) The method of claim 20, wherein the anorexic drug is sibturamine or fenfloruamine or lepin.

22. (Previously presented) The method of claim 1, wherein the paradigm is based on animals, which are fed, fasted or sated.

23. (Previously presented) The method of claim 1, wherein in the paradigm, the subjects having differential levels of protein expression comprise normal subjects and underweight or overweight subjects.

24. (Cancelled)

25. (Original) The method of claim 24, wherein the differential levels of protein expression are not observed in normal subjects who have and have not been treated with the drug which reduces body weight ~~agent~~.

26. (Currently amended) The method of claim 1, wherein in the paradigm, the subjects having differential levels of protein expression comprise:

(a) normal subjects who have and have not been treated with the drug which reduces body weight ~~agent~~; and

(b) subjects, having at least one of body weight or eating disordered function, who have and have not been treated with the drug which reduces body weight ~~agent~~.

27. (Previously presented) The method of claim 26, wherein the differential levels of protein expression are not observed in normal subjects and subjects having at least one of body weight or eating disordered function, both subject groups being untreated with the drug which reduces body weight ~~agent~~.

28. (Previously presented) The method of claim 1, wherein the paradigm is established using two-dimensional gel electrophoresis carried out on the relevant tissue or a protein-containing extract thereof.

29. (Previously presented) The method of claim 1, further comprising the step of isolating a differentially expressed protein identified in the method.

30. (Original) The method of claim 29, further comprising the step of characterising the isolated protein.

31. (Previously presented) The method of claim 1, wherein the at least one differentially expressed protein comprises at least one of MOM 31, MOM 32, MOM 34, MOM 36, MOM 33, MOM 35, WOM 37, WOM 52, WOM 60, WOM 38, WOM 53, WOM 61, WOM 44, WOM 54, WOM 62, WOM 45, WOM 55, WOM 63, WOM 48, WOM 57, WOM 64, WOM 49, WOM 58, WOM 50, WOM 59, WOM 39, WOM 40, WOM 41, WOM 42, WOM 43, WOM 46, WOM 47, WOM 51, BOM 66, BOM 67, BOM 68, BOM 75, BOM 76, BOM 77, BOM 69, BOM 70, BOM 71, BOM 72, BOM 73, BOM 74.

32. (Previously presented) The method of claim 30, further comprising performing an assay in which the protein is used to determine its specific binding partners.

33. (Previously presented) The method of claim 30, further comprising performing an assay in which the protein is used to screen for its agonists or antagonists.

34. (Previously presented) The method of claim 1, wherein the agents are screened using a high throughput screening method.

35. (Previously presented) A method of making a pharmaceutical composition which comprises, after having identified an agent using the method of claim 1, the further step of manufacturing the agent and formulating it with an acceptable carrier to provide the pharmaceutical composition.

36. (Original) A protein for use in a method of medical treatment wherein the protein is selected from MOM 31, MOM 32,

MOM 34, MOM 36, MOM 33, MOM 35, WOM 37, WOM 52, WOM 60, WOM 38, WOM 53, WOM 61, WOM 44, WOM 54, WOM 62, WOM 45, WOM 55, WOM 63, WOM 48, WOM 57, WOM 64, WOM 49, WOM 58, WOM 50, WOM 59, WOM 39, WOM 40, WOM 41, WOM 42, WOM 43, WOM 46, WOM 47, WOM 51, BOM 66, BOM 67, BOM 68, BOM 75, BOM 76, BOM 77, BOM 69, BOM 70, BOM 71, BOM 72, BOM 73, BOM 74.

37-39 Cancelled

40. (Previously presented) A method of treating a condition characterised by at least one body weight or eating dysfunction in a patient, the method comprising administering a therapeutically or prophylactically effective amount of an agent identified by a method of claim 1 to the patient.

41. (Previously presented) The method of claim 40, wherein the at least one body weight or eating dysfunction is a result of obesity, non-insulin dependent diabetes or type 2 diabetes, anorexia nervosa, bulimia or cachexia induced by AIDS or cancer or trauma.

42. (Previously presented) A method of determining the nature or degree of at least one body weight or eating dysfunction in a human or animal subject, the method comprising:

(a) establishing a paradigm in which at least one protein is differentially expressed in relevant tissue from, or representative of, subjects having differential levels of at least one body weight or eating function;

(b) obtaining a sample of the tissue from the subject;

(c) determining the presence, absence or degree of expression of the at least one differentially expressed protein in the sample; and

(d) relating the determination to the nature or degree of the body weight or eating dysfunction by reference to a previous correlation between such a determination and clinical information.

43. (Original) The method of claim 42, wherein the sample is a tissue sample or body fluid sample or urine.

44. (Previously presented) The method of claim 42, wherein in the paradigm at least four proteins are differentially expressed, providing a multi-protein fingerprint of the nature or degree of the body weight or eating dysfunction.

45. (Previously presented) The method of claim 42, which further comprises determining an effective therapy for treating the body weight or eating dysfunction.

46-47 Cancelled

48. (Previously presented) A method of claim 59, wherein the body weight or eating dysfunction state is obesity.

49. (Previously presented) A protein which is differentially expressed in relevant tissue from, or representative of subjects having differential levels of body weight or eating dysfunction and which is obtainable by the method of two-dimensional gel electrophoresis carried out on said tissue or a protein-containing extract thereof, the method comprising:

(a) providing non-linear immobilized pH gradient (IPG) strips of acrylamide polymer 3 mm x 180 mm;

(b) rehydrating the IPG strips in a cassette containing 25 ml. of an aqueous solution of urea (8M), 3-[(cholamidopropyl)dimethylammonio]-1-propanesulphonate (CHAPS, 2% w/v), dithioerythritol (DTE, 10mM), mixture of acids and bases of pH 3.5 to 10 (2% w/v) and a trace of Bromophenol Blue;

(c) emptying the cassette of liquid, transferring the strips to an electrophoretic tray fitted with humid electrode wicks, electrodes and sample cups, covering the strips and cups with low viscosity paraffin oil;

(d) applying 200 micrograms of an aqueous solution of dried, powdered material of the relevant body tissue in urea (8M), CHAPS (4% w/v), Tris (40 mM), DTE (65 mM), SDS (0.05% w/v) and a trace of Bromophenol Blue to the sample cups, at the cathodic end of the IPG strips;

(e) carrying out isoelectric focusing on the gel at a voltage which increases linearly from 300 to 3500 V during 3 hours, followed by another 3 hours at 3500 V, and thereafter at 5000V for a time effective to enable the proteins to migrate in the strips to their pI-dependent final positions;

(f) equilibrating the strips within the tray with 100 ml of an aqueous solution containing Tris-HCl (50 mM) pH 6.8, urea (6M), glycerol (30% v/v), SDS (2% w/v) and DTE (2% w/v) for 12 minutes;

(g) replacing this solution by 100 ml. of an aqueous solution containing Tris-HCl (50 mM) pH 6.8, urea (6M), glycerol (30% v/v), SDS (2% w/v), iodoacetamide(2.5% w/v) and a trace of Bromophenol Blue for 5 minutes;

(h) providing a vertical gradient slab gel 160 x 200 x 1.5 mm of acrylamide/piperazine-diacrylyl cross-linker(9-16%T/2.6%C), polymerised in the presence of TEMED (0.5% w/v), ammonium persulphate (0.1% w/v) and sodium thiosulphate (5 mM), in Tris-HCl (0.375M) pH 8.8 as leading buffer;

(i) over-layering the gel with sec-butanol for about 2 hours, removing the overlay and replacing it with water;

(j) cutting the IPG gel strips to a size suitable for the second dimensional electrophoresis, removing 6 mm from the anode end and 14 mm from the cathode end;

(k) over-layering the slab gel with an aqueous solution of agarose (0.5% w/v) and Tris-glycine-SDS (25 mM-198 mM-0.1% w/v) as leading buffer, heated to 70°C and loading the IPG gel strips

onto the slab gel through this over-layered solution;

(l) running the second dimensional electrophoresis at a constant current of 40 mA at 8-12°C for 5 hours; and

(m) washing the gel.

50. (Original) The protein of claim 49, wherein the protein is selected from MOM 31, MOM 32, MOM 34, MOM 36, MOM 33, MOM 35, WOM 37, WOM 52, WOM 60, WOM 38, WOM 53, WOM 61, WOM 44, WOM 54, WOM 62, WOM 45, WOM 55, WOM 63, WOM 48, WOM 57, WOM 64, WOM 49, WOM 58, WOM 50, WOM 59, WOM 39, WOM 40, WOM 41, WOM 42, WOM 43, WOM 46, WOM 47, WOM 51, BOM 66, BOM 67, BOM 68, BOM 75, BOM 76, BOM 77, BOM 69, BOM 70, BOM 71, BOM 72, BOM 73, BOM 74.

51. (Original) A differentially expressed protein having one or more of the identifying characteristics as set out in any one of Tables 3 to 5.

52. (Original) The protein of claim 51, wherein the identifying characteristics are pI and Mw.

53. (Previously presented) The method of claim 9, wherein said genetic mutation is at least one of ob/ob, db/db agouti, fat, tub and fa/fa.

54. (Previously presented) The method of claim 12, wherein said desert rodents are from the group of spiny mice or sand rats.

55. (Previously presented) The method of claim 31, further comprising performing an assay in which the protein is used to determine its specific binding partners.

56. (Previously presented) The method of claim 31, further comprising performing an assay in which the protein is used to screen for its agonists or antagonists.

57. (Previously presented) The method of claim 29, wherein the proteins are screened using a high throughput screening method.

58. (Previously presented) A method of preventing the redevelopment of obesity in body weight reduced previously obese subjects, said method comprising treating said subjects with an agent that will restore the expression of at least one differentially expressed protein in the body weight or eating dysfunction state to that found in the normal state.

59. (Previously presented) A method for the prediction of the most appropriate and effective treatment to alleviate body weight or eating dysfunction state in a patient and to monitor the success of said treatment, said method comprising determining a pattern of differentially expressed proteins in a tissue sample or body fluid sample or urine of a patient with body weight or eating dysfunction; and making said prediction on the basis of said pattern.